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Typhoid Fever in Infants under Two Years of Age

ETIOLOGY—PATHOLOGY—SYMPTOMATOLOGY,

BY

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TYPHOID FEVER

IN

INFANTS UNDER TWO YEARS OF AGE.

ETIOLOGY—PATHOLOGY—SYMPTOMATOLOGY.¹

IF an apology is necessary for introducing the subject of typhoid fever into this Society, after the recent thorough discussion of the entire subject in the Medical Society of the District of Columbia, it is to be found in the fact that the disease is so rarely met with in infants, or at least is so rarely diagnosticated by the post-mortem appearances, that we feel justified in presenting it here. We have been induced to select this subject owing to a discussion of the same subject which took place in the American Pediatric Society two years ago, in which Dr. Northrup, of New York, claimed that this disease did not occur in infants, or, at least, that it had not been found in the pathological examinations made in the New York Foundling Asylum; and that "swollen Peyer's patches and mesenteric glands and spleen in children cannot safely be interpreted like similar lesions in adults." He further says: "So many undefined fevers in infantile life drag along, variously diagnosticated; so many undefined cases come to autopsy, revealing swollen Peyer's patches, swollen mesenteric lymph nodes, enlarged spleen—what shall we say of them? The

¹ Read before the Washington Obstetrical and Gynecological Society.



symptoms of typhoid and no autopsy; the autopsy of typhoid and no symptoms; bacterially no typhoid bacilli."

In the discussion which followed this paper the consensus of opinion seemed to be that typhoid did exist in infancy, and I took the ground that, though I had not been able to verify my diagnosis by the typical anatomical lesions, nevertheless I was certain that I had treated the train of symptoms which clinically characterize this disease in the adult.

Previous to 1840 it was believed that infancy and early childhood were immune against this disease. Prior to this date the disease seems to have been defined under the term "infantile remittent fever." There can be no question that it existed prior to this date and that the mistake was in not differentiating it from other febrile diseases, though about this date Stewart, in his work, clearly defines the disease as occurring in infancy; still even much earlier it was described by Abercrombie in a 6-months-old girl and in another 7 months old, the latter being confirmed by a necropsy. West in 1822 described the disease fully from a clinical and anatomical standpoint. Millard in 1828 reported two cases in children. Characla in 1840 reported a child 8 days old who died; it presented enlargement of the follicles, Peyer's patches, and mesenteric glands. Shadler narrates the case of a 7-months-old child whose mother died on the twenty-sixth day of typhoid fever. Five days after its mother's death the infant sickened, and died on the eleventh day; ulceration and infiltration of Peyer's patches, swollen mesenteric glands, and enlargement and softening of the spleen were found. Earl, of Chicago, reported a case 24 months old that had the typical symptoms of typhoid, complicated by intestinal hemorrhage, in which the necropsy revealed the characteristic local lesions. Numerous other cases under 2 years of age have been cited. Not only have infants contracted it from their mothers at birth, but a fetus of seven months is reported as having it.

Etiology.—A tangible cause has unquestionably been found for this disease, and the specific bacillus is found in the lesion. The poison does not originate spontaneously from decomposing animal or vegetable matter, but must have been transported from some infected individual. So far as infants are concerned the proof of the presence of the bacillus is still negative. Eberth claims to have found the bacilli in the tissues of a fetus of the

twentieth week's gestation, as well as in the intervillous spaces of the placenta, and developed cultures from them. The theory of the transformation of the germs outside of the body—that is, the transformation of the bacillus coli communis into the bacillus of Eberth—is still *sub judice*. Although this point has not been definitely determined, we must depend upon the experimental inoculation of animals for its settlement. Water is probably a good carrier of the disease in infancy, though milk, which readily takes up bacilli of other varieties, may be the common carrier.

Pathology.—The same anatomical lesions exist in the child and in the infant that are found in the adult. It will not be long before the presence of the bacilli will be demonstrated in the lymph structures of the bowel, spleen, and other organs. Gerhart believes that the lesions in children differ from those in the adult, especially in the beginning. The swelling of Peyer's patches shows itself earlier, and is seen with greater frequency near the ileo-cecal valve, though it may reach higher in the small intestine. The swollen patches often project above the mucous membrane, and above them are often seen denudation and slight ulceration. From these sites the destructive processes extend. It has been suggested that this difference in the amount of destruction may be due to the character of the food of the infant, which is fluid and non-irritating. In the case which I present below, the anatomical lesions in the intestines seem to be as typical as those in the adult.

Clinical History.—As far as the clinical picture is concerned, there does not seem to be any marked difference in the child and adult, but in the infant there is unquestionably a difference. Restlessness is marked and the fever persists for days with only slight irritation of the gastro-enteric tract. The temperature, which usually reaches a higher range than in the adult, is well borne, and it is surprising how long some infants can bear a continuous high temperature. If headache is present it is not recognized. There is usually nothing characteristic in the appearance of the tongue. Vomiting has been observed in a few cases, but is probably due to forcing food. The appetite is uncertain, sometimes voracious, at others almost entirely absent. Constipation is usually present throughout the entire course of the disease making it necessary to relieve the bowel by enemata. The typical rose spots are not always present or are seldom recognized. Tympany is rare. Hemorrhage from the bowel is sel-

dom seen, though some observers mention it. Peritonitis may or may not be present. The spleen, if enlarged, is seldom detected, and Northrup claims that it cannot be recognized unless it project below the margin of the ribs. The liver is probably unaffected. The kidneys are not affected. Epistaxis is rare. Bronchitis has been observed in a few cases, but is probably due to hypostatic congestion. Relapses occur in a fair percentage of cases.

The following case is unquestionably one of typhoid fever. The diagnosis was not positively made until the necropsy. The specimen was examined and pronounced to be typhoid fever. Subsequently the report of the bacteriologist, though not confirmatory, did not deny that it was the disease.

Florence P., aged 2 years, mulatto, was admitted to the Children's Hospital, D. C., September 19th, 1893.

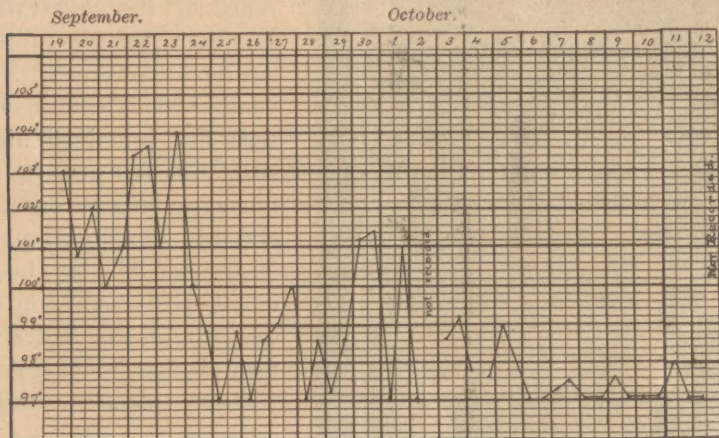
Previous History.—Her father has consumption, but her mother's health is fair.

Child was well until one month ago, when she was taken with diarrhea which lasted a few days. About one week ago there was another attack of diarrhea, accompanied by nausea, loss of appetite, and languor. At times the stools contained blood and mucus, but were unaccompanied by pain and straining. She has had a slight cough for several days.

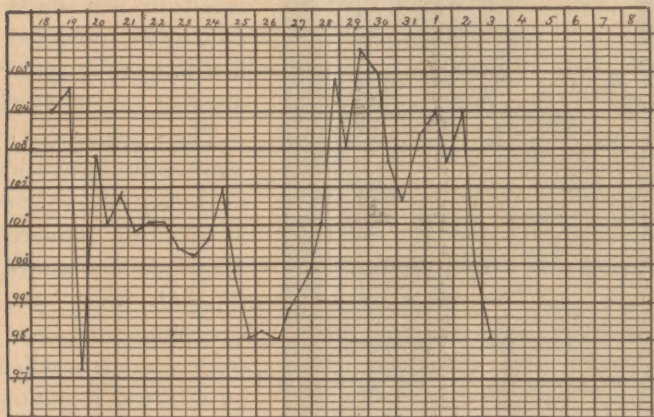
Present Condition.—The child's general appearance indicated severe illness, but the emaciation, high temperature, and rapid pulse, with the frequent muco-sanguinolent stools, led us to suspect an enteritis catarrhalis. She was placed upon milk diet; sponge baths to reduce the temperature; and in a few days the diarrhea ceased. (See chart.)

September 23d: Paralysis of extensor muscles of left hand noted. Paralysis extended gradually until the 29th, when the extensors of both hands and feet were paralyzed. September 30th: Upon returning to duty, after my vacation, I found the foregoing notes upon this case. The temperature had now gone up to 101.2°, and there was a persistent tonic contraction of flexors of both upper and lower extremities. Efforts to extend the limbs caused the child to scream, but she was so stiff that she could be rolled from side to side without discomfort. Irritability was the only mental disturbance noted. Fluid extract of ergot, gtt. iiij. every three hours, was given, and by October 10th the rigidity had entirely disappeared, she was eating and

digesting well and was rapidly convalescing. Medicinal treatment was now discontinued. October 19th: While sitting in her carriage yesterday the patient was suddenly seized with violent jerky movements of upper extremities. She was restless, and the temperature was noted to be 104° . This morning



October.



Temperature Chart.

it reached 104.6° , and phenacetin and calomel were ordered. One grain of phenacetin in two doses having reduced the temperature more than 7° , it was discontinued. October 20th: Temperature 102.8° and rigidity of extremities marked. Sulphate of quinine, gr. i. by suppository, every three hours during the day. October 26th: The rigidity has disappeared and the



Ileo-colic portion of intestine, showing thickening and ulceration of solitary bodies and Peyer's patches of ileum.

temperature has remained subnormal for twenty-four hours, so the quinine is to be stopped. October 28th: There is a recurrence of the aggravated symptoms, so the suppositories are resumed. November 2d: The temperature has remained high since the last report, so the suppositories are ordered to be discontinued. November 3d: Has been in collapse since noon of the 2d, and has had sixteen profuse liquid stools, without blood. Death occurred at 2 P.M.

During the last four weeks of her illness constipation prevailed and had to be relieved by enemata or mild purgatives.

The irregularity of the temperature wave and the evidences of cerebro-spinal irritation rendered the diagnosis so obscure that typhoid fever was not suspected until a few days prior to death, when the quinine was found to have little or no effect upon the hyperpyrexia.

Necropsy, six hours after death.—*Brain*: Marked congestion of entire brain, more on the right side; left hemisphere covered with a gluey

substance which filled the sulci, especially abundant around Sylvian fissure. *Heart*: Normal. *Lungs*: Marked hypostatic congestion. *Abdomen*: Liver normal; gall bladder empty and pale; spleen enlarged, congested, and softened; kidneys normal. *Stomach*: Congested and contained about a pint of grumous material, which was not examined chemically or microscopically; mesenteric glands enlarged and softened. *Intestines* (macroscopic) contained a quantity of yellow, watery feces. The lower end of ileum shows thickening and ulceration of Peyer's patches (see cut), and to a less extent of the solitary follicles. The solitary follicles of the cecum are also ulcerated.

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